Intradural Lumbar Disc Herniation: A Rare Case

İntradural Lomber Disk Hernisi: Nadir Bir Olgu Sunumu

Intradural Lomber Disk Hernisi / Intradural Lumbar Disc Herniation

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VI. Sinir Sistemi Cerrahisi Kongresinde (2010) poster bildiri olarak sunulmuştur.

Özet

65 yaşında erkek hasta bel ve her iki bacakta ağrı, idrar-gayta yapamama, erektil disfonksiyon ile başvurdu. Lomber MRG tetkikinde T2 ağırlıklı görüntülerde L4-5 seviyesinde kanal içini dolduran ve posterior longitudinal ligament (PLL) devamlılığını bozan ekstrüde disk hernisi saptandı. Ameliyatta dural kese açıldığında granülasyonla örtülü disk fragmanları saptandı ve tamamen boşaltıldı. Ameliyat sonrası ağrıları geçen hastanın 3 ay sonrası kontrolünda idrar-gayta yapmasının tama yakın düzeldiği ve erektil disfonksiyonun ortadan kalktığı saptandı. Lomber MRG tetkikinde nüks yada psödomeningosel gelişimi saptanmadı. Bu olgudan da anlaşıldığı üzere MRG tetkikleri incelendiğinde iki durumun IDH şüphesini artırabileceği düşünülmüştür. Birinci nokta PLL devamlılığının olmaması, ikinci nokta ise herniye olmuş intervertebral diskin spinal kanala gaga şeklinde uzanım göstermesidir. Bu hastalarda prognozun herniye diskin tamamen boşaltılması ve semptomların süresi ve karakteri ile yakın ilişkili olduğu saptanmıştır.

Anahtar Kelimeler

Cauda Equina Sendromu; Intradural Disk; Durotomi

Abstract

A 65-year-old man was admitted with radiating pain in right leg and saddle type anaesthesia with urination, defecation and ejaculation problems. Spinal MRI revealed a disc fragment that hugely extruded towards central spinal channel with marked cranial migration at L4-5 level on the T2 weighted sagittal image. It also demonstrated abrupt loss of continuity of the posterior longitudinal ligament (PLL). Then, he was taken for surgery. After a durotomy was performed, two pieces of cartilagenous tissue were removed en bloc. Three months later, his urination, defecation, ejaculation returned to nearly normal; and postoperative MRI revealed that operative site was clean and there was no residual disc material. We would like to emphasis on two points of MR findings about which increase the suspicious for intradural extension of these disc fragments. The first point is abrupt loss of continuity of the posterior longitudinal ligament. The second point is a sharp beak-like appearance on T2 weighted axial imaging. Prognosis is related to complete removal of the herniated material, cleaning of the intervertebral space, duration and characterization of the symptomatology.

Keywords

Cauda Equina Syndrome; Intradural Disc; Durotomy

DOI: 10.4328/JCAM.639 Received: 04.03.2011 Accepted: 30.03.2011 Printed: 01.05.2013 J Clin Anal Med 2013;4(3): 233-5
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Introduction

Intradural lumbar disc herniation (IDLDH) is a rare condition (0.04-0.33%), and its pathogenesis has not been clearly understood. Most of the authors reported that adhesion between the dura mater and posterior longitudinal ligament causing from previous spine surgery has been accepted as a predisposing factor [1]. Here we report an adult patient with IDLDH.

Case Report

A 65-year-old man was admitted to our clinic with a history of extreme back pain while standing still and/ or any movement. Straight leg-raising test was positive on both sides. There was no real power or sensorial deficit. Plain spine X-rays were found out normal. Magnetic resonance imaging (MRI) disclosed a huge disc herniation extended towards the central spinal channel at L4-5 level (Figure 1). After he had been operated because of

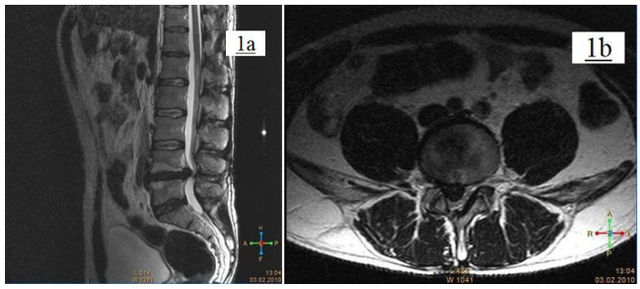

Figure 1. On first admission of the patient, preoperative MR scans (A-B) demonstrated disc herniation at L4-5 level. T2-weighted images revealed a disc fragment that hugely extruded toward to central spinal channel

these symptoms and findings at another medical center, he was not able to urinate, defecate and ejaculate. And four months later, he was admitted to our center again. On this second admission, he had also radiating pain in right leg and saddle type anaesthesia. Spinal MRI revealed a disc fragment that hugely extruded towards the central spinal channel with marked cranial migration at L4-5 level on the T2 weighted sagittal image (Figure 2). It also demonstrated abrupt loss of continuity

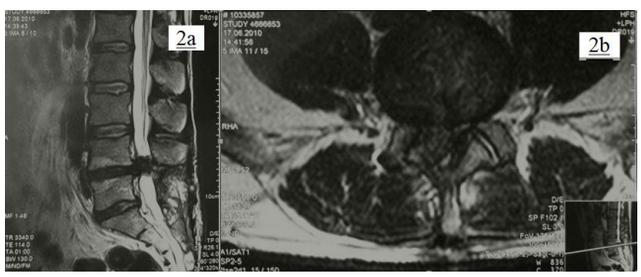

Figure 2. On second admission of the patient, the MR scans (A-B) revealed a disc fragment that hugely extruded toward to central spinal channel with marked cranial migration at L4-5 level on the T2 weighted sagittal image. It also demonstrated abrupt loss of continuity of the posterior longitudinal ligament.

of the posterior longitudinal ligament (PLL). So, he was taken to emergent surgery. Through a total L4 laminectomy and spacious bilateral L5 foraminatomy, spinal cord and L5 roots were found immobile and upon gentle palpation, a tense dura could be felt with abnormal swelling of the dural sac. At this point, a durotomy was performed and bilobulated-yellowish encapsulated mass which was compressing the rootlets and displacing them toward the right side within the dural sac was seen. When its capsule was punctured two pieces of cartilagenous tissue which also contained end plates were removed en bloc. After removing the disc fragments, a hole was discovered in the ventral aspect of the dura. The dural tissue around the hole was firmly adhered to the PLL. After the dorsal aspect of the dura mater was closed with 4/0 silk suture, the epidural space was explored on both sides of the dural sac and protruded L4-5 intervertebral disc was emptied. Surgical materials were sent for histopathological examination (Figure 3). Histopathological diagnosis was degenerated cartilagenous tissue.

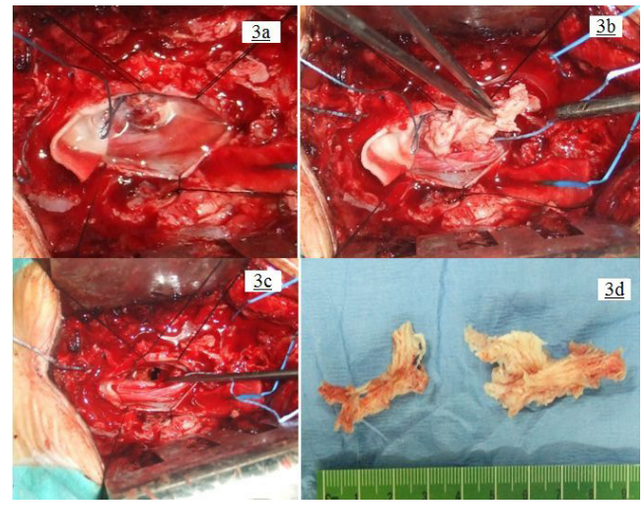

Figure 3. After durotomy (A-B) bilobulated-yellowish encapsulated mass which was compresing the rootlets and displacing them towards the right side within the dural sac was seen; and when its capsule was punctured two pieces of cartilagenous tissue was removed en bloc. (C-D) After removing the disc fragments a hole was discovered in the ventral aspect of the dura mater.

The patient was discharged with 90/90 Karnofsky Performance Score. The postoperative early course was uneventful. Three months later, his urination, defecation, ejaculation became nearly normal; and postoperative MRI revealed that operative site was clean and there was no residual disc material (Figure 4).

Discussion

IDLDH is a rare condition and it usually occurs at the L4-5 level (55%), L3-4 level (16%), and L5-S1 level (10%) [1].

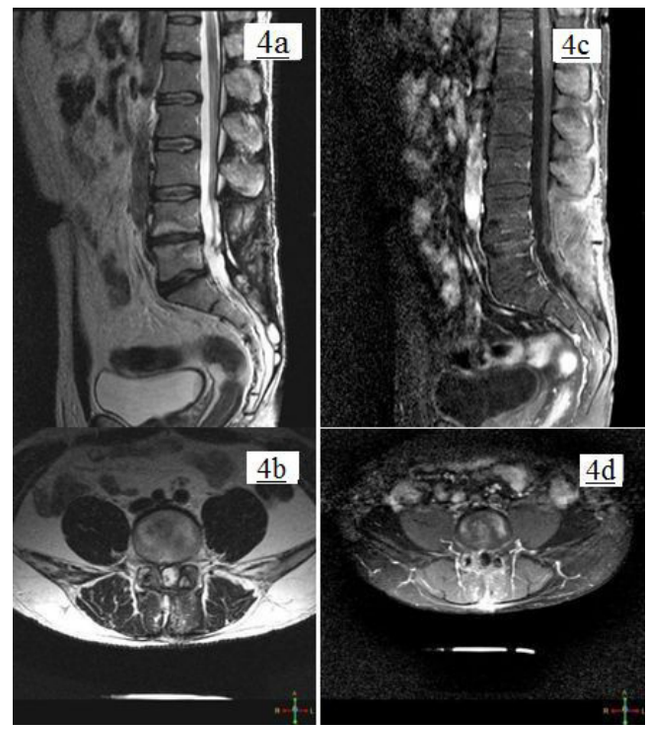

Figure 4. Postoperative T2 WI MR scan (A-B) and MR scan with gadolinium (C-D) revealed that operative site was clean and there was no residual disc material.

The mechanism of intradural disc herniation is unclear; but some predisposing anatomic factors may contribute to it as follows:
- Adhesions between the annulus fibrosus, PLL and dura mater.
- Congenital narrowing of the spinal canal with less epidural space.
- Congenital or iatrogenic fineness of the dura mater [2].

The existence of adhesions between the ventral wall of the dura mater and PLL causing from previous surgery was demonstrated in most of the cases. These adhesions may be produced by the local inflammatory processes, congenital union between the dura mater and PLL, traumatic irritation such as previous surgery or herniated disc [3]. Our patient had a previous disc surgery history at the same side which was performed at a different medical center. We could not obtain the MR images of this first postoperative period. For this reason, we could not discuss if our patient's disc fragments was a consequence of adhesion between the ventral dura mater and PLL which was developed after this previous surgery or not. But after removal of the disc fragments, there was a hole which was strongly adhered to the intervertebral space by granulomatous tissue. Because of the presence of this hole surrounded with granulomatous tissue, we thought that this huge disc fragment might be most probably missed out, or it was not removed totally from the intradural space at the first operation. Koç et al. (2001) and Choi et al (2007) suggested that closure of the ventrally located dural tear is necessary because of the risk of CSF leakage [3, 4]. Although, we used no barrier material (such as cyanoacyrlate applying, fat tissue packing or primary repairing the dural defect) to close this hole, CSF fistula was not observed at the surgical area neither intra- nor post-operative period. All these findings also support our thesis mentioned above.

IDLDH should be distinguished from neurinoma, meningioma, ependymoma, infections, epidermoid, and dermoid tumours. To distinguish intradural disc herniation can be made by using of the myelography, CT, CT-myelography, MRI, MR-myelography. CT usually does not provide a specific appearance of intradural herniation because of the bony artefacts. However, intradural mass with the same density as that of the intervertebral disc, and no enhancement after contrast medium administration can help diagnosis of intradural herniation. Preoperative diagnosis of IDLDH can also be made by a MRI with gadolinium. Acute IDLDH does not show the enhancement, but chronic IDLDH shows ring enhancement in T1WI after injection of the contrast medium which is attributed to vascular granulation tissue around the lesion. However, almost in all cases described in literature, diagnosis of IDLDH could be identified only during surgery [2]. We agree with Choi et al (2007) that two points of MRI findings may cause us to suspect for intradural extension of these disc fragments. The first point is abrupt loss of continuity of the posterior longitudinal ligament (PLL). The second point is a sharp beak-like appearance on T2 weighted axial imaging [3]. Anal sphincter disease and cauda equina syndrome have an incidence of 30% of all reported cases in literature. In about 67% of the patients are able to go back to work without neurologic deficit, while 33% of cases have residual neurologic deficit such as micturition urgency, sensory loss, and muscular weakness. Prognosis is related to complete removal of the herniated material, cleaning of the intervertebral space, duration and characterization of the symptomatology (such as cauda equina syndrome), and previous surgery of the lumbar spinal column. In 62% of the patients with cauda equina syndrome, the prognosis is associated with slow but full recovery from deficits taking a minimum 3 weeks to a maximum of 32 months. In 38% of the patients, prognosis is related to incomplete resolution with the persistence of perineum sensory loss and atrophy anal sphincter and erectile dysfunction [2]. Three months later, the urination, the defecation, and the ejaculation functions of our patients became nearly normal.

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